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LETTERS TO THE EDITOR

Regarding 'Liver cell adenoma and liver cell adenomatosis' by Ludger Barthelmes and Iain S. Tait

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Sir,

We read with great interest the recent article by Barthelmes and Tait (*HPB* 2005;7:186–196).

The impact of the diagnosis 'liver cell adenoma' on the life of a young female patient is enormous. Conservative management of liver cell adenoma frequently implies cessation of oral contraceptives, regular follow-up including radiological imaging, and negative advice regarding pregnancy. Surgical treatment of solitary adenomas implies the risk of morbidity and mortality following hepatic resection and does not guarantee relief of complaints. However, the risk of bleeding from or malignant transformation of that solitary adenoma is prevented. The debate whether to manage solitary adenomas conservatively or surgically continues. A recent retrospective study by Toso et al. [1] recommended resection of solitary adenomas. The review by Barthelmes and Tait summarized most of the insight in the diagnosis and management of liver cell adenomas during the past decades. Based on their review, they strongly recommended surgical resection for hepatocellular adenomas.

The investigators reviewed 14 adenoma series that included 167 patients in total and covered the era between 1979 and 2002. The authors addressed the diagnostic difficulties, in particular the differentiation of liver cell adenomas from well-differentiated hepatocellular carcinoma. Furthermore, they discussed the risk of bleeding and malignant transformation. Although we agree with the authors that hepatic lesions with an uncertain diagnosis or adenomas at risk for bleeding or malignant transformation should be treated surgically, we think conservative treatment including careful follow-up is applicable to some patients.

Our current management of liver cell adenoma is based on the study by Terkivatan et al. [2], which included 33 patients, of whom 19 were treated surgically and 14 were treated conservatively. The guideline to excise liver cell adenomas if larger than 5 cm, referenced by Barthelmes and Tait, originated from that study. With this guideline, we saved several women from hepatic resection. In the past 5 years, we discussed all patients with benign liver tumours in a weekly multidisciplinary meeting with a hepatologist, a radiologist specialized in the liver and a pathologist. Conservative management was chosen in case of a certain benign radiological diagnosis and a lesion smaller than 5 cm. In case of absent regression, adenomas were resected at a later stage. After withdrawal from oral contraceptives, bleeds did not occur.

We believe that this management is justified due to the rapid evolution of magnetic resonance imaging (MRI) of benign liver tumours. Further prospective studies that include modern MRI approaches should determine whether lesions with a certain radiological diagnosis of liver cell adenoma can also progress to a carcinoma or that the reported cases of malignant degeneration in the past originated from lesions which could not possibly be characterized as either liver cell adenoma or hepatocellular carcinoma. Modern molecular techniques can possibly be helpful in distinguishing the two different entities [3].

Furthermore, it is not well documented whether bleeds occur after cessation of oral contraceptives. The main problem of most of the cited studies is the retrospective design. Prospective studies addressing

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the superiority of surgical management over conservative management in patients with a clear diagnosis of solitary liver cell adenoma have to be conducted before conservative treatment can be abandoned.

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Ascariasis-induced eosinophilic cholecystitis – a unique case

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Sir,

I would like to bring to the attention of your readers the following unique case of ascariasis-induced eosinophilic cholecystitis. The condition was first described in 1949 and is characterized by dense, transmural infiltration of the gall bladder wall with eosinophils [1]. This rare condition has received attention from French workers [2] who consider it to be a discrete clinical and pathological entity due to a local allergic process within the gall bladder. On occasion, a large number of eosinophil leukocytes are seen in the wall of surgically removed gall bladders. It is recognized that eosinophils may appear in and often from a substantial component of the pleomorphic cellular infiltrate which is found in gall bladders removed 2–3 weeks after an episode of acute cholecystitis, but the significance of an almost pure eosinophilia of the gall bladder is usually not clear either to the histopathologist or the surgeon. Eosinophilic infiltration of the biliary tract may be idiopathic or may represent a variant of eosinophilic gastroenteritis or may be associated with parasitic infestation. Histopathologically there should be little problem in differentiating eosinophilic cholecystitis from the more usually encountered varieties, clinically there seems to be no difference. The presence of eosinophilia in certain parasitic infections, particularly those helminthic infections that invade tissue, has been recognized particularly from the time the eosinophil was discovered. Yet its role in parasitic disease is still disputed. Although eosinophils are capable of phagocytosing bacteria and other microorganisms, their efficacy in this regard is felt to be inferior to the neutrophil.

A 40-year-old female presented to our hospital with signs and symptoms of acute cholecystitis. Abdominal ultrasonography revealed a distended thick-walled gall bladder with a long coiled tubular echogenic structure within it. A diagnosis of gall bladder ascariasis with acute cholecystitis was made. The patient was managed conservatively but did not show any improvement even after 1 week's treatment. Cholecystectomy was carried out and on histopathological examination of the specimen a pure transmural infiltration of numerous eosinophils in the gall bladder tissue was seen and a final diagnosis of eosinophilic cholecystitis was made. This is the first reported case of eosinophilic cholecystitis induced by gall bladder ascariasis. The parasite may have induced a hypersensitivity reaction with numerous eosinophils in the gall bladder wall. In a recent report of eosinophilic cholecystitis associated with hepatic hydatid cyst ruptured into biliary tract, the cause of eosinophilic cholecystitis was similarly attributed to the hypersensitivity reaction [3]. We would agree with the French workers [2] who consider this to be a local allergic process that

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